Dizziness

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KEYWORDS

• Dizziness • Vertigo • Ataxia • HINTS • BPPV • Dix-Hallpike • Epley • Stroke

KEY POINTS

- A structured problem-based history and physical examination using the ATTEST and STANDING algorithms are essential for accurate diagnosis and treatment for patients with dizziness.
- Initially look for signs of stroke including dysarthria, dysphagia, dysphonia, dysmetria, diplopia, acute hearing loss, acute hearing loss, or an abnormal NIH stroke scale or obvious medical causes.
- Use the Dix-Hallpike maneuver or supine roll maneuver to diagnose benign positional paroxysmal vertigo.
- Use the HINTS examination (peripheral head impulse test, unilateral horizonal nystagmus, no vertical skew deviation) to diagnose acute vestibular neuritis in patients with acute vestibular syndrome.
- Noncontrast head computed tomography is very low yield in the assessment of the acutely dizzy patient.

Video content accompanies this article at http://www.emed.theclinics.com.

INTRODUCTION

Dizziness accounts for 2.1% to 3.6% of annual emergency department (ED) visits and an estimated $10 billion in health care costs in the United States. Diagnosing dizziness presents unique challenges due to its broad differential.[1] While most causes are benign, ischemic stroke is an important cause. In an effort to "rule out" serious pathology, clinicians often rely on head computed tomographies (CTs), which add cost, prolong ED stays, and provide false reassurance rather than diagnostic clarity.[1] A structured, problem-based history and physical examination are essential for accurate diagnosis and treatment.

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Abbreviations	
AICA	anterior inferior cerebellar artery
AVS	acute vestibular syndrome
BPPV	benign paroxysmal positional vertigo
CPPV	central paroxysmal positional vertigo
CT	computed tomography
ED	emergency department
EVS	episodic vestibular syndrome
hc	horizontal canal
HIT	head impulse test
ICH	intracranial hemorrhage
pc	posterior canal
PICA	posterior inferior cerebellar artery
s-EVS	spontaneous episodic vestibular syndrome
t-EVS	triggered episodic vestibular syndrome
VM	vestibular migraine
VOR	vestibulo-ocular reflex

ANATOMY, PHYSIOLOGY, AND PATHOPHYSIOLOGY

The vestibular system has both peripheral (semicircular canals, utricle, and saccule—see Fig. 1) and central (ocular system, brainstem, cerebellum, cortex, and postural muscles) components.[2] Disruption in any parts of this system can cause dizziness. In the ED, dizziness often stems from nonvestibular causes such as pneumonia, pulmonary embolism, fever, hypoglycemia, acute coronary syndrome, dysrhythmia, or medication effects. These conditions can indirectly affect the vestibular system. Peripheral (inner ear) and central (brain) causes, however, have more defined mechanisms. Understanding vestibular anatomy and physiology aids in a systematic, problem-based approach to dizziness.

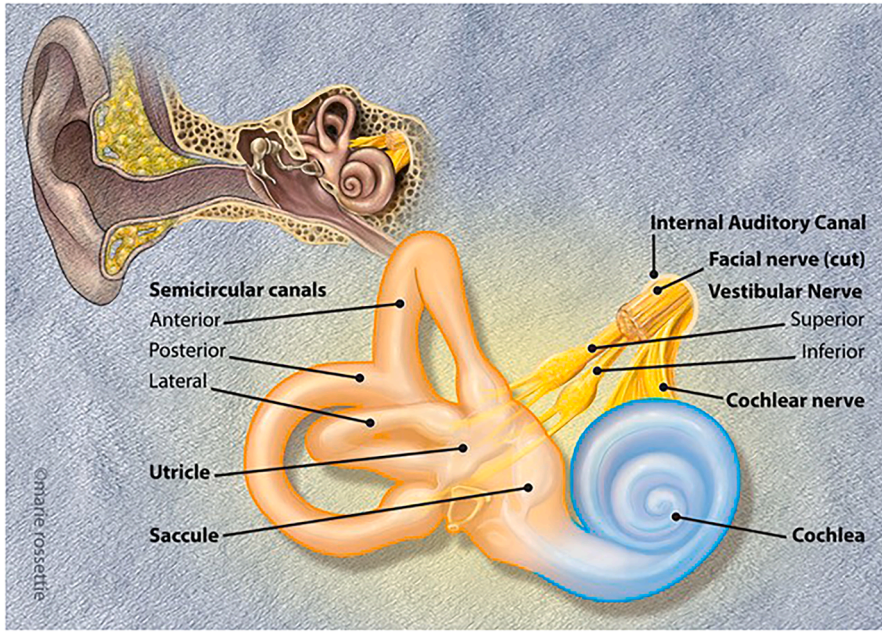


Fig. 1. Inner ear anatomy. (*Image Courtesy by* Marie Rossettie.)

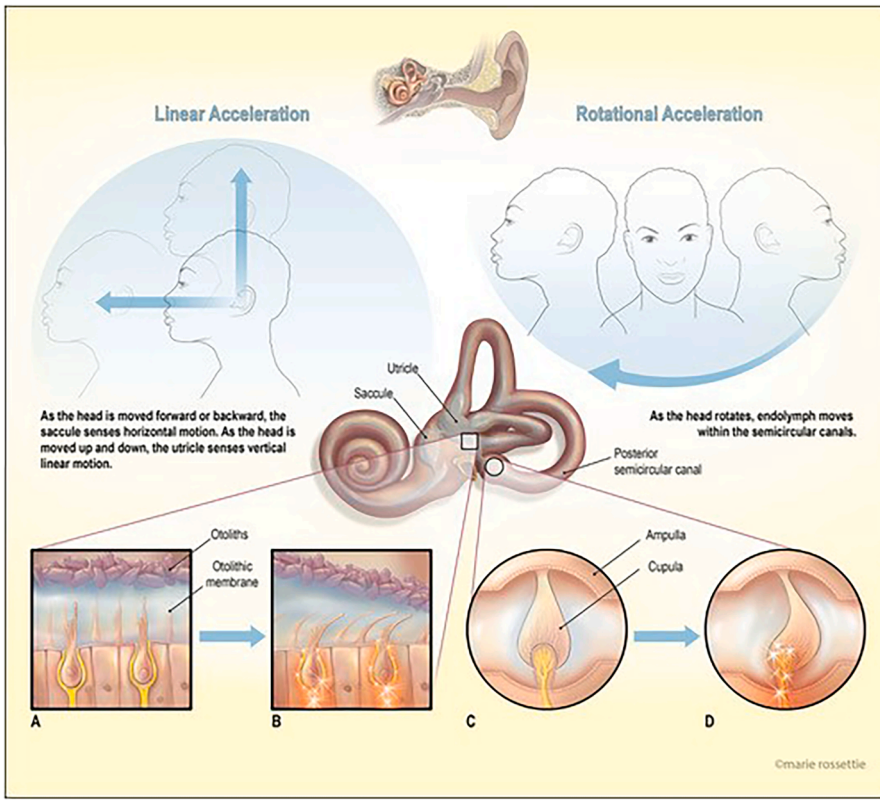


Fig. 2. Inner ear physiology. (*Image Courtesy by* Marie Rossettie.)

The inner ear (labyrinth) consists of the peripheral vestibular apparatus and the cochlea. The 3 paired semicircular canals detect rotational motion and the utricle and saccule sense linear motion (**Fig. 2**). These structures contain endolymph, a fluid that moves in response to head motion. The utricle and saccule are lined with mechanoreceptors (hair cells) embedded in a gel-like membrane containing calcium carbonate crystals (otoliths). At the end of each semicircular canal is the ampulla, where the crista ampullaris—coated by the thicker cupula—detects rotational movement as fluid displacement deflects the cupula.[2]

Benign paroxysmal positional vertigo (BPPV) occurs when dislodged otoliths from the utricle migrate into a semicircular canal, most commonly the posterior canal, which causes the brain to perceive nonexistent motion.[3] Most commonly, the otoliths are free-floating in the semicircular canal (**Fig. 3**, Panel A) but in some cases, they become stuck on the cupula (see **Fig. 3**, Panel B).

The eighth cranial nerve comprises the vestibular and cochlear nerves. Signals from the vestibular labyrinth travel via the vestibular nerve to the brainstem's vestibular nuclei, which relay information to the cerebellum, oculomotor system, cortex, and spinal cord. The vestibulo-ocular reflex (VOR) stabilizes gaze during head movement. The horizontal head impulse test (HIT), first described in 1988, identifies VOR failure due to vestibular nerve or nucleus dysfunction.[4] Since this reflex arc bypasses the cerebellum, the VOR is affected in vestibular neuritis (inflammation of the vestibular nerve) but not in cerebellar strokes.

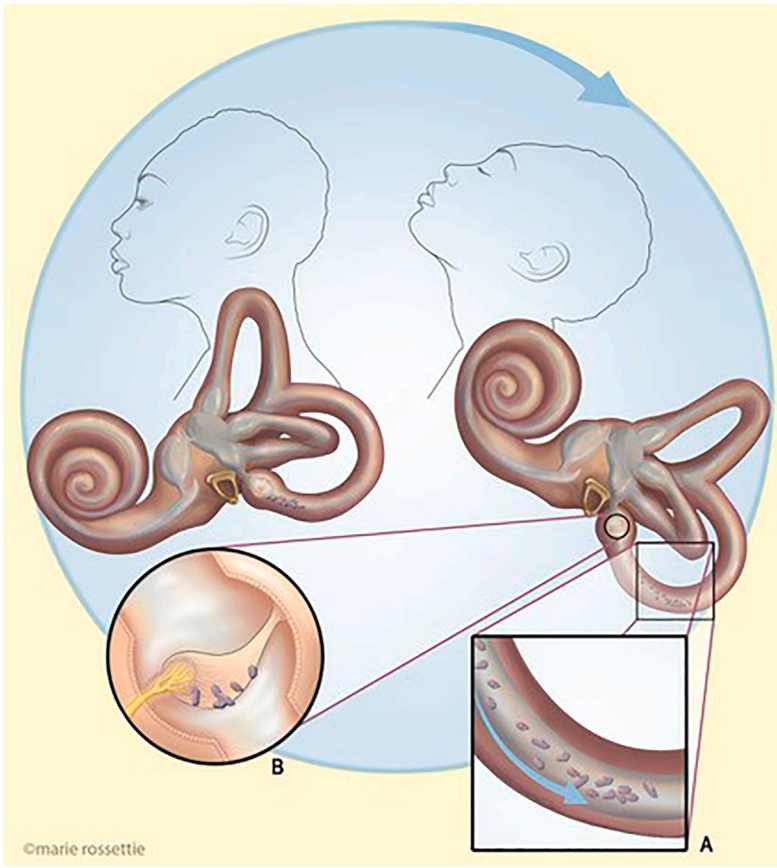


Fig. 3. Pathophysiology of BPPV. (*Image Courtesy by* Marie Rossettie.)

The posterior circulation is supplied by the vertebral arteries, which join to form the basilar artery. Before merging, they give rise to the posterior inferior cerebellar artery (PICA). Two other paired cerebellar arteries, the anterior inferior cerebellar artery (AICA), and the superior cerebellar artery arise from the basilar artery. The PICA supplies the lateral medulla and inferior cerebellum, while the AICA supplies the lateral pons, including the vestibular nerve root entry zone. A branch of AICA, the labyrinthine artery, supplies the peripheral labyrinth. Strokes affecting AICA can cause labyrinthine or vestibular nucleus ischemia, mimicking vestibular neuritis and leading to a falsely reassuring horizontal impulse test (HIT) (**Fig. 4**).

DIFFERENTIAL DIAGNOSIS AND MISDIAGNOSIS

The causes of dizziness are numerous. In an analysis from a large National Hospital Ambulatory Medical Care Survey database[5] of ED patients, the proportion of causes of dizziness listed in the charts were:

- General medical conditions: 49%
- Otologic or vestibular conditions: 33%
- Cardiovascular causes: 21%

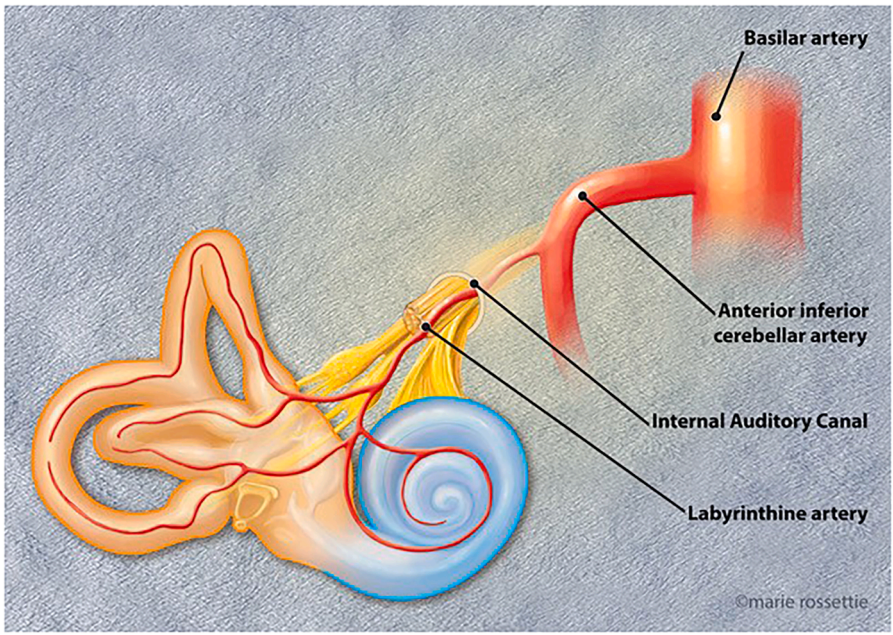


Fig. 4. AICA and its branches. (*Image Courtesy by* Marie Rossettie.)

- Respiratory conditions: 12%
- Neurologic diseases: 7%
- Cerebrovascular causes: 4%

Misdiagnosis of patients with acute dizziness, especially of cerebellar and brainstem stroke is common. In a German study of 475 ED patients with dizziness, all assessed by a neurologist in the ED, nearly 50% of diagnoses were changed by a second neurologist (blinded to the initial diagnosis) on follow-up.[6] Evolution of the clinical course over time after the initial evaluation was a factor in 70% of the misdiagnosed patients.[6] Most posterior circulation strokes that present primarily with dizziness do not score high on the National institute of Health Stroke Scale.[7] Posterior strokes are misdiagnosed more than twice as often as anterior ones.[8] Posterior stroke is a very rare presentation in ED patients who present with isolated dizziness. In one study of 1666 adult ED patients with dizziness, fewer than 1% of those with *isolated* dizziness had a cerebrovascular cause.[9] Other studies found that of ED patients with dizziness who are discharged with a peripheral vestibular diagnosis, only 0.14% to 0.5% are subsequently hospitalized with a stroke.[10–13] Although these proportions are low, because dizziness is so common, the absolute number of patients involved is significant.

Retrospective studies report that between 28% and 59% of patients with cerebellar strokes are missed in the ED.[14,15] Factors associated with incorrect diagnosis include lack of understanding of how to interpret nystagmus, and false reassurance of younger patient age, absent traditional risk factors, and negative noncontrast head computed tomography (CT).[8,16,17]

Given how uncommon strokes presenting as isolated dizziness are, clinicians may go their entire career and only see a few patients with strokes presenting as isolated dizziness and thus, without adequate follow-up, they may be falsely reassured of their clinical diagnosis and that their diagnostic process is sound.[18]

A negative CT has very low sensitivity in ischemic strokes presenting as isolated dizziness and can falsely reassure clinicians who initially had a diagnostic concern. In a study of ED patients with dizziness who were discharged with a benign ICD-9 "dizzy" diagnosis and followed-up for 30 days, patients who returned with a stroke were 2.3 times more likely to have had a CT on the first visit, suggesting that physicians were correctly determining worrisome patients but then applying the wrong diagnostic test.[19]

DIAGNOSTIC APPROACH

A systematic approach that first rules out obvious strokes or non-neurologic causes, followed by targeted history and physical examination maneuvers, is more effective and cost-efficient.[20,21]

The evidence-based society of academic emergency medicine (SAEM) guidelines for reasonable and approriate care in the emergency department (GRACE-3) committee's recommendations are based on patients' timing and triggers presentation.[20] This is the ATTEST algorithm which is rooted in the history and physical examination (**Fig. 5**). In a national survey, 78% of Emergency Medicine Residency Program directors supported teaching HINTS.[22] In isolated acute vestibular syndrome (AVS) cases, imaging is not recommended if HINTS is performed by trained providers and is consistent with a peripheral cause.[23]

Although not validated in routine ED use, ATTEST aligns with standard diagnostic approaches and current evidence.[20]

STEP 1: RULE OUT STROKE

Look for "Deadly D" signs—dysarthria, dysphagia, dysphonia, dysmetria, diplopia—as well as acute hearing loss, severe ataxia, or abnormal NIH stroke scale findings. If present, activate the local stroke protocol.

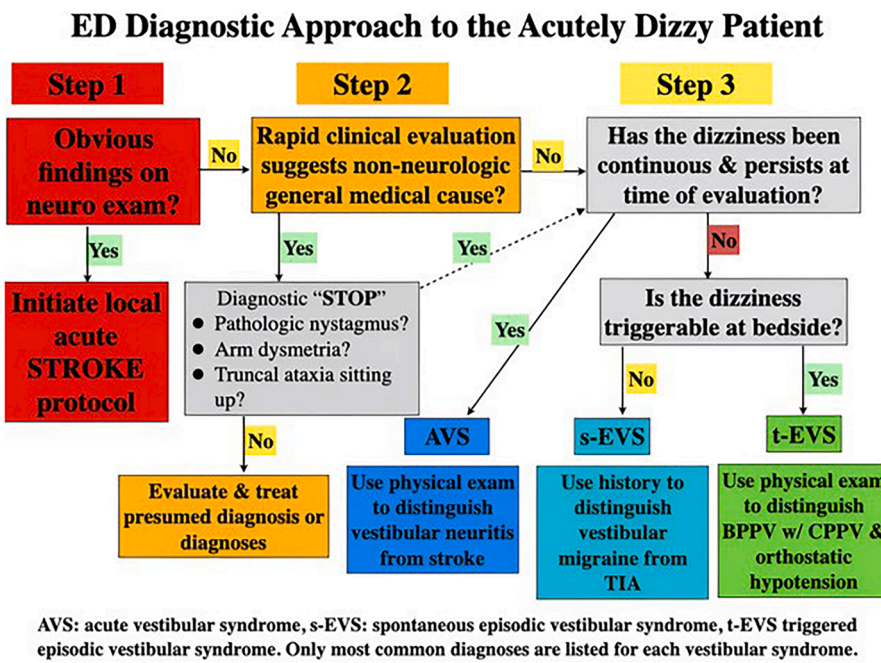

ED Diagnostic Approach to the Acutely Dizzy Patient
Step 1
Step 2
Step 3
Obvious findings on neuro exam?
No
Rapid clinical evaluation suggests non-neurologic general medical cause?
No
Has the dizziness been continuous & persists at time of evaluation?
Yes
Yes
Yes
No
Initiate local acute STROKE protocol
Diagnostic "STOP"
Pathologic nystagmus?
Arm dysmetria?
Truncal ataxia sitting up?
Yes
Is the dizziness triggerable at bedside?
No
Yes
No
AVS
s-EVS
t-EVS
Evaluate & treat presumed diagnosis or diagnoses
Use physical exam to distinguish vestibular neuritis from stroke
Use history to distinguish vestibular migraine from TIA
Use physical exam to distinguish BPPV w/ CPPV & orthostatic hypotension
AVS: acute vestibular syndrome, s-EVS: spontaneous episodic vestibular syndrome, t-EVS triggered episodic vestibular syndrome. Only most common diagnoses are listed for each vestibular syndrome.

Fig. 5. ATTEST algorithm. * Each is the final part of the algorithm.

Table 1
General medical causes of dizziness

General Medical Causes of Dizziness	History Suggestive of Diagnosis
GI bleed	Black stools, tachycardia, hypotension
Medications/illicit drugs and alcohol	New anticonvulsants, antihypertensives
Trauma	Recent head injury
Pregnancy-related	Abdominal pain, vaginal bleeding, positive pregnancy test
Metabolic	Abnormal glucose, Wernicke's
Cardiac/pulmonary	Chest pain, dyspnea, tachycardia, hypoxia
Febrile illnesses	Fever, symptoms related to specific infection

STEP 2: IDENTIFY MEDICAL CAUSES

Around 50% of dizziness cases stem from general medical conditions. These are usually suggested by co-chief complaints and vital sign abnormalities. See **Table 1** for examples.

If a medical diagnosis is likely, perform an STOP check: is there pathologic nystagmus, is there arm dysmetria on finger-to-nose testing, or is there truncal ataxia (have patient sit up unaided and close eyes)? If STOP is reassuring, treat the presumed condition; if worrisome, proceed to Step 3, which places the patient into one of 3 timing and triggers categories, each with its own differential diagnosis and physical examination elements to sort through that differential diagnosis.

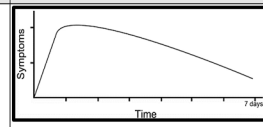
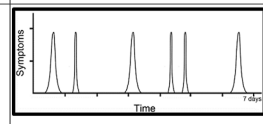
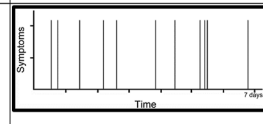
Syndrome	Description	Graphic representation of symptoms over time	Common benign causes	Common Serious Causes
Acute Vestibular Syndrome (AVS)	A clinical syndrome of acute -onset continuous dizziness lasting days to weeks and generally including features suggestive of new, ongoing vestibular system dysfunction (e.g. Nausea and vomiting, nystagmus and postural instability. In the ED, they are symptomatic even when at rest		Vestibular neuritis Labyrinthitis	Posterior circulation ischemic stroke
Episodic Syndromes	Clinical syndromes of multiple, recurrent attacks of dizziness, which many be spontaneous or triggered. If the patient presents during a first attack and is actively symptomatic, approach as acute vestibular syndrome			
Spontaneous Episodic Vestibular Syndrome (s-EVS)	A clinical syndrome of transient dizziness usually lasting minutes to hours and generally including nausea and vomiting, postural instability and nystagmus. There are usually multiple recurrent attacks with repeated spells but may initially present after a first attack. There are no clear trigger of these attacks. In the ED, these patients are generally asymptomatic at rest		Vestibular Migraine Meniere's disease	Transient ischemic attack
Triggered Episodic Vestibular Syndrome (t-EVS)	A clinical syndrome of brief transient dizziness lasting seconds to minutes and generally including nausea and vomiting, postural instability. There are usually multiple recurrent attacks with repeated spells but some patients may initially present after a first attack. These attacks have a clear positional trigger, usually movement of the head or standing up. In the ED, these patients are generally asymptomatic at rest but the symptoms can be triggered at bedside		BPPV	CPPV Orthostatic hypotension due to serious medical illness

Fig. 6. Different categories of vestibular syndrome.

STEP 3: ACUTE VESTIBULAR SYNDROME VERSUS EPISODIC VESTIBULAR SYNDROME

Patients with continuous dizziness have an AVS, 75% are due to neuritis, 20% due to stroke, and 2% to 3% are caused by multiple sclerosis (**Fig. 6**).[24] Another uncommon, but important, treatable, cause is Wernicke encephalopathy (thiamine deficiency).[25]

ACUTE VESTIBULAR SYNDROME

Patients with the *AVS* experience sudden or rapid onset dizziness usually lasting days, and which persists even at rest without head motion. However, symptoms often *worsen* with head movement. The belief that dizziness that worsens with head motion indicates a peripheral cause is a common misconception.[26] Vestibular neuritis is presumably caused by viral or postviral inflammation, similar to a Bell's Palsy. Most cases are idiopathic, possibly linked to herpes simplex infections.[27] Ramsay Hunt syndrome due to herpes zoster presents with AVS, usually with hearing loss, facial palsy, and a vesicular eruption in the external ear canal or palate.[28] However, posterior fossa strokes can mimic vestibular neuritis or labyrinthitis.[27]

The prevalence of cerebrovascular disease in patients presenting to the ED with dizziness is 3% to 5%[1] but among those with an AVS it is estimated at approximately 20% to 25%.[27] Almost all (96%) of these strokes are ischemic.[27,28] Fortunately, physical examination findings can reliably distinguish between vestibular neuritis and posterior circulation stroke with greater sensitivity than an early MRI.[29–31]

KEY EXAMINATION STEPS FOR ATTEST ACUTE VESTIBULAR SYNDROME

1. Is there central-type nystagmus or *absent* nystagmus (**Fig. 7**)?
2. Is vertical skew deviation present? (Horizontal corrections are *not* skew deviation)

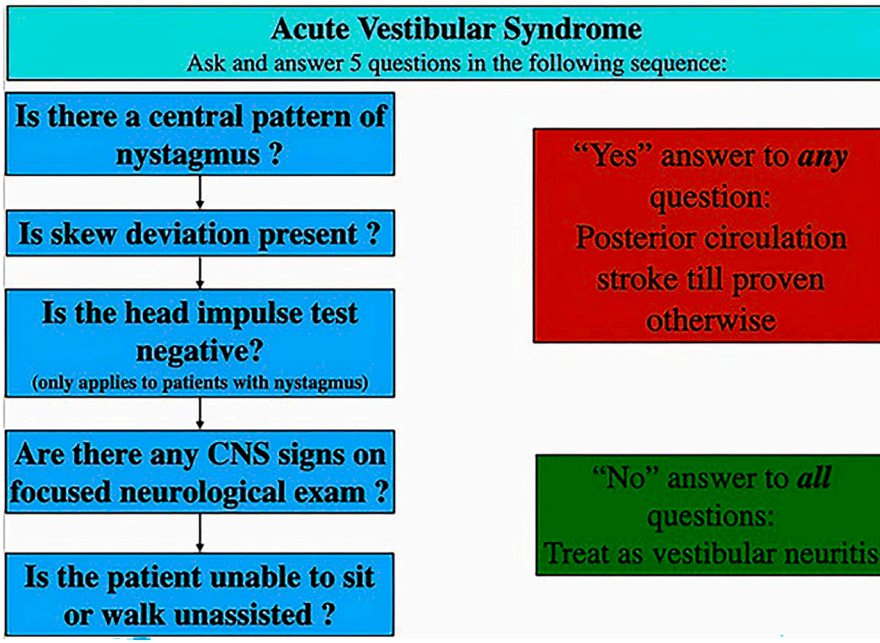

Acute Vestibular Syndrome
Ask and answer 5 questions in the following sequence:
Is there a central pattern of nystagmus ?
Is skew deviation present ?
Is the head impulse test negative?
(only applies to patients with nystagmus)
Are there any CNS signs on focused neurological exam ?
Is the patient unable to sit or walk unassisted ?
"Yes" answer to any question: Posterior circulation stroke till proven otherwise
"No" answer to all questions: Treat as vestibular neuritis

Fig. 7. Distinguishing peripheral from central causes of an AVS.

3. Is HIT worrisome (no corrective saccade)?
4. Are CNS findings present on a posterior circulation examination?
5. Can the patient sit/walk unassisted?

A "Yes" to any suggests a central cause (likely stroke) → hospital admission.[27–29]

NYSTAGMUS IN ACUTE VESTIBULAR SYNDROME

Understanding the details of nystagmus is fundamental. We recommend testing nystagmus first because it is easy for the patient to do, helps define the differential diagnosis and indicates if the HIT should be used or not. In a study of 1091 dizzy patients in US EDs, physicians used templates to document the presence or absence of nystagmus in 887 (80%).[30] Nystagmus was documented as present in 185 (21%), of which diagnostically meaningful information was recorded in only 10 (5.4%). In patients given a peripheral diagnosis, the nystagmus description conflicted with that diagnosis in 81%.[30] By convention, nystagmus is named for the direction of the fast phase. Direction-changing or gaze evoked nystagmus, when the fast phase changes direction (fast beating to the right on rightward gaze and left beating on leftward gaze) is always caused by a central process. Nearly, all patients with vestibular neuritis have unidirectional horizontal nystagmus.[31] Thus, the absence of nystagmus in a patient with an AVS is worrisome.[3] Similarly, nystagmus that is vertical, direction-changing, or torsional also indicate a central cause. Nystagmus may be absent or dampened if vestibular suppressant medications (eg, benzodiazepines) are used prior to the evaluation. Normal visual fixation can completely suppress mild nystagmus in patients with vestibular neuritis.[32]

If unidirectional horizontal nystagmus is absent, acute vestibular neuritis is unlikely to be the etiology, and a central cause must be pursued. Gait testing is especially useful in this group of patients—an AVS without nystagmus.

Special one-way goggles called Frenzel lenses block visual fixation that improves the detection of nystagmus. If a clinician does not have Frenzel lenses, one can use a penlight to do the same. Shine a penlight into one eye while testing the other.[32] Using a sheet of white paper can also reduce fixation and uncover mild or hard-to-see nystagmus. Place the paper in front of the patient and look around the sheet while testing eye movements.[33]

SKEW DEVIATION

Skew deviation is a vertical misalignment of the eyes due to imbalance in gravity-sensing vestibular pathways. Skew deviation is elicited using the "alternate cover" test. With the patient looking directly at the examiners nose, the physician rapidly covers one eye, then the other, and continues alternating back and forth. If skew deviation is present, each time the covered eye is uncovered, a slight vertical correction occurs. Focus your attention on one eye only (it does not matter which eye) to increase the likelihood of your seeing this subtle finding. The presence of skew deviation should always be considered a central finding.

THE HEAD IMPULSE TEST

The VOR stabilizes vision by keeping the retina focused on an object while the head moves. The left and right vestibular nuclei normally discharge at equal rates. When one nucleus (or the vestibular nerve) is affected, unidirectional horizontal nystagmus develops (fast component *away* from the affected side), and a horizontal catch-up saccade occurs when the head is moved *toward* the affected side (**Fig. 8**).

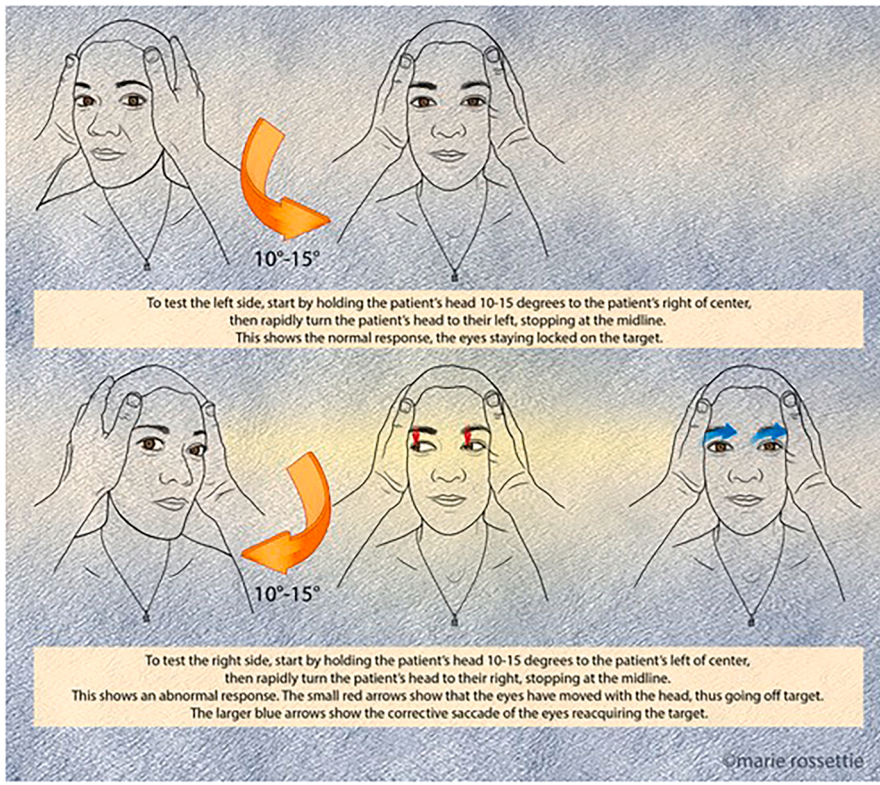

10°-15°
To test the left side, start by holding the patient's head 10-15 degrees to the patient's right of center,
then rapidly turn the patient's head to their left, stopping at the midline.
This shows the normal response, the eyes staying locked on the target.
10°-15°
To test the right side, start by holding the patient's head 10-15 degrees to the patient's left of center,
then rapidly turn the patient's head to their right, stopping at the midline.
This shows an abnormal response. The small red arrows show that the eyes have moved with the head, thus going off target.
The larger blue arrows show the corrective saccade of the eyes reacquiring the target.
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Fig. 8. The head impulse test. (*Image Courtesy by* Marie Rossettie.)

The HIT assesses VOR disruption.[2] In vestibular neuritis, an inflamed vestibular nerve causes a refixation delay (corrective saccade) upon rapid head movement. *The HIT is only meaningful in patients with an AVS plus nystagmus!*

Standing in front of the patient, the clinician holds the patients head by each side, instructs the patient to focus on the clinician's nose and keep head and neck loose. The clinician moves the head 10° to 15° from midline then *very rapidly* (>120°/s) brings it to the center position and stop at midline. In normal vestibular function, the patient's gaze remains locked on the examiner's nose. The presence of a corrective saccade (the eyes move with the head, then snap back refix on the clinician's nose) usually indicates vestibular neuritis but false positives occur in patients with AICA or labyrinthine artery strokes. If there is no corrective saccade, a central process must be considered. The only reported complication of the HIT is a case of transient, spontaneously resolving complete heart block from the vagal stimulation.[34]

Because the presence of a refixation saccade is a "positive" test, but is usually reassuring for a peripheral process, it is best to report or document what actually happens or what the conclusion is, rather than HIT (or HINTS) positive or negative. For example, "the HIT (or HINTS) shows a corrective saccade on leftward motion" or "the HINTS examination is consistent with a peripheral finding" (Videos 1 and 2).[35]

ADDITIONAL CENTRAL NERVOUS SYSTEM EXAMINATION

Because many clinicians are not yet familiar with the HIT or with HINTS, we recommend examining the structures served by the posterior circulation—cranial nerves, cerebellum, and occipital lobes. New central nervous system (CNS) findings strongly suggest a central cause of dizziness. A Horner's syndrome (ptosis, miosis, anhidrosis) can be seen with a lateral medullary infarct.[36]

Testing hearing which is the "plus" of HINTS +, was added as a fourth component to detect those patients with AICA and labyrinthine strokes.[27] Between 35% and 75% of patients with an AVS plus hearing loss have stroke as a cause.[36,37] Over half of patients with a new hearing loss did not perceive it, highlighting the importance of testing hearing.

GAIT TESTING

Patients unable to walk should not be discharged. In a study of 114 AVS patients, most with neuritis could walk independently, while stroke patients could not (**Table 2**).[38]

A recent meta-analysis looked at the diagnostic accuracy of physical examinations in vertigo patients (**Table 3**).[39]

SPONTANEOUS EPISODIC VESTIBULAR SYNDROME (INTERMITTENT, SPONTANEOUS)

Patients with spontaneous episodic vestibular syndrome (s-EVS) have brief (minutes to hours), untriggered episodes of dizziness and are not dizzy at the time of ED evaluation. Because s-EVS patients are asymptomatic and their dizziness cannot be triggered, a thorough history is needed as physical examination is not helpful. If a patient *is symptomatic* at presentation, the approach would be the same as for an AVS. The most common cause of s-EVS is vestibular migraine (VM) and the most serious is posterior circulation transient ischemic attack (TIA). Other causes include panic attacks, vasovagal syncope, Meniere's disease, arrhythmia, myocardial infarct (MI), pulmonary embolism (PE), hypoglycemia, and carbon monoxide (CO) poisoning.[1] VM is often misdiagnosed. VM diagnosis requires 5 episodes with vestibular symptoms, migraine

Table 2
Assessment of gait unsteadiness[38]

Severity of Gait Unsteadiness	Definition	Positive Predictive Value of Ataxia Grade for Stroke
Grade 0	Normal	0% (n = 0/5) with no unsteadiness had stroke
Grade 1	Mild to moderate imbalance with walking independently	7% (n = 3/42) with Grade 1 unsteadiness had stroke
Grade 2	Severe imbalance when standing or cannot walk without support	28% (n = 11/39) with Grade 2 unsteadiness had stroke
Grade 3	Falling at upright posture/inability to stand unaided	100% (n = 28/28) with Grade 3 unsteadiness had stroke

Adapted from Carmona S, Martínez C, Zalazar G, et al. The diagnostic accuracy of truncal ataxia and HINTS as cardinal signs for acute vestibular syndrome. Front Neurol 2016;7. https://doi.org/10.3389/fneur.2016.00125.

Table 3
Meta-analysis of the diagnostic accuracy of physical examinations in acute vertigo[39]

Physical Examination Maneuver	Sensitivity %	Specificity %
Truncal/gait ataxia	69.7	83.7
Spontaneous nystagmus	52.3	42
Nystagmus type (central)	50.7	98.5
Test of skew	23.7	98.5
HIT	76.8	89.1
HINTS (HIT, horizontal nystagmus, test of skew)	92.9	83.4
HINTS+ (HINTS + Hearing loss)	99.0	84.8

From Shah VP, e Silva LOJ, Farah W, et al. Diagnostic accuracy of the physical examination in emergency department patients with acute vertigo or dizziness: A systematic review and meta-analysis for GRACE-3. Acad Emerg Med 2023;30(5):552–78. https://doi.org/10.1111/ACEM.14630.

history, and migraine-like features in at least half. Symptoms last seconds to days. Headache may or may not accompany the dizziness.[40,41]

The most serious cause of the spontaneous EVS is central posterior circulation TIA should be suspected in patients with high ABCD2 scores. A recent review highlights the distinction with VM.[42] Consider cardiac arrhythmias in unexplained dizziness without a trigger.

TRIGGERED EPISODIC VESTIBULAR SYNDROME

Triggered episodic vestibular syndrome (t-EVS) causes brief episodes of vertigo (seconds to minutes) triggered by movement, sound, or Valsalva.[1] Symptoms should be triggerable at the bedside. The most common causes are BPPV and orthostatic hypotension. Clinicians should check for orthostatic hypotension in patients with positional triggers.[1] Central paroxysmal positional vertigo (CPPV) from posterior fossa lesions, strokes, must be distinguished from BPPV, as it presents with unique nystagmus (see Video 3).[35] CPPV is rare in an "all-comer" ED population.

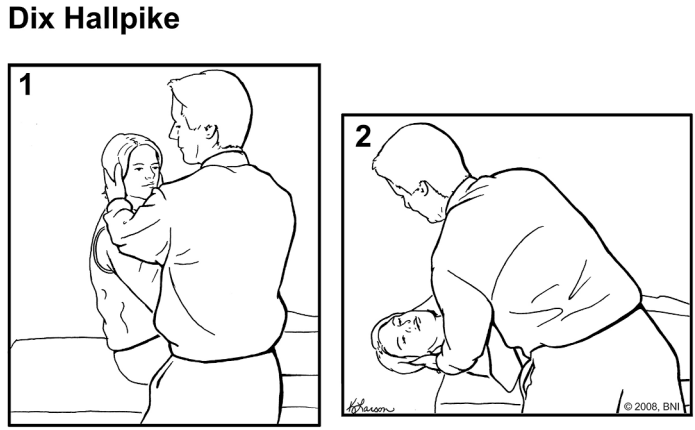

Fig. 9. Diagnosing posterior canal BPPV with the Dix-Hallpike (*Used with permission from* Barrow Neurological Institute, Phoenix, Arizona.)

One important and common clue to BPPV, which distinguishes it from orthostatic hypotension is having episodes while supine, especially at night while in bed.[43,44] One study of dizzy patients reported a positive odds ratio for BPPV of 60 for dizzy episodes that occurred during sleep.[43] Importantly, some patients with BPPV will feel a vague sense of lingering dizziness in between episodes, but this is different from the severe episodes.

For suspected BPPV, bedside testing is diagnostic (**Fig. 9**). The posterior canal (pc-BPPV) is assessed with the Dix-Hallpike maneuver, while the horizontal canal (hc-BPPV) is tested with the supine roll test. Some hc-BPPV patients may exhibit spontaneous horizontal nystagmus due to the nature of the horizonal canal being in plane while seated or standing, unlike the usual triggered nystagmus of BPPV.[45]

In the Dix-Hallpike maneuver, the patient starts in the seated position with the head turned 45° toward the side being tested, and then is quickly laid down and the affected ear is placed down resulting in upbeat torsional nystagmus. See Video 4.[35]

In some patients whose history suggests BPPV, the Dix-Hallpike shows clear unilateral symptoms but no nystagmus.[46–48] In these cases, treatment is often successful (**Fig. 10**, Video 5).[33]

Although traditionally considered far less common than pc-BPPV, a recent large analysis reported that hc-BPPV was nearly as common as pc-BPPV.[49] The symptoms can be evoked using the supine roll test (see **Fig. 10**). Lie the patient supine with their head 30° from horizontal. Rotating to the right provokes horizontal nystagmus that changes direction depending on which side the head is being turned toward. Because anterior canal BPPV is rare and because downbeat torsional nystagmus if often due to serious central causes, exercise extreme caution in diagnosing ac-BPPV.[35]

Standing Algorithm

The *STANDING*[50] algorithm has been shown to be as effective for emergency physicians as for neuro-otologists.[21] It is similar to ATTEST and uses the HINTS examination but omits the test of skew (see image). STANDING requires Frenzel Lenses, which

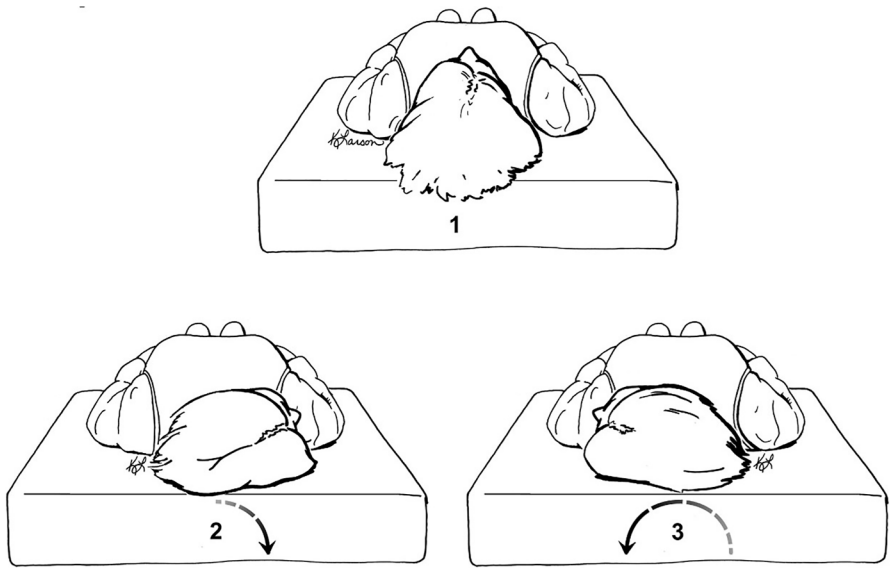

Fig. 10. Diagnosing horizontal canal BPPV with the supine roll test.

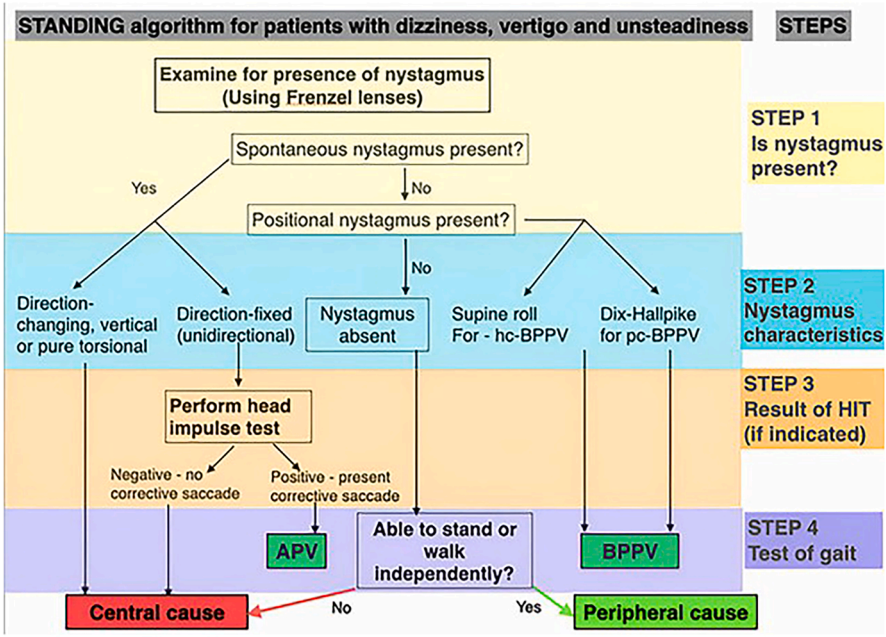

Fig. 11. STANDING algorithm.

enhances detection of peripheral nystagmus. Advantages of the STANDING protocol over HINTS is that it includes diagnosis of BPPV and forces examination of the gait, and does not require patients to be placed into a specific timing and triggers category (Fig. 11).

See the below image.

Diagnostic studies

If a medical cause is suspected, pursue the underlying etiology (eg, check blood glucose in altered mental status with dizziness).

CT is rarely indicated and can lead to false reassurance.[19] In 2 large ED studies, 0% (0/344) and 1.6% (7/448) of dizzy patients had emergent CT findings.[51,52] Intracranial hemorrhage (ICH) rarely presents as isolated AVS, but CT may be justified if headache is prominent, or anticoagulation is present. Early MRI can miss strokes within 48 hours. In a meta-analysis of 3236 patients, 7% had false-negative DWI-MRI, especially in posterior circulation strokes.[53] False negatives range from 12% to 18% in AVS (first 72 hours)[53–55] and approach 50% in strokes less than 10 mm.[55] While delayed MRI (>72 hours) is the gold standard, as earlier, the physical examination, if done properly, is extremely sensitive to determine a central etiology.

For s-EVS, physical examination does not reliably distinguish VM from posterior TIA—decisions must be individualized, but this is one instance where noninvasive angiography such as CT angiography may be useful.[40–42,56–58]

For t-EVS (likely BPPV), physical examination suffices. A therapeutic canalith repositioning maneuver may serve as a diagnostic test.

Treatments

Posterior canal benign paroxysmal positional vertigo. While there are several different therapeutic maneuvers, the treatment usually involves using an Epley maneuver to

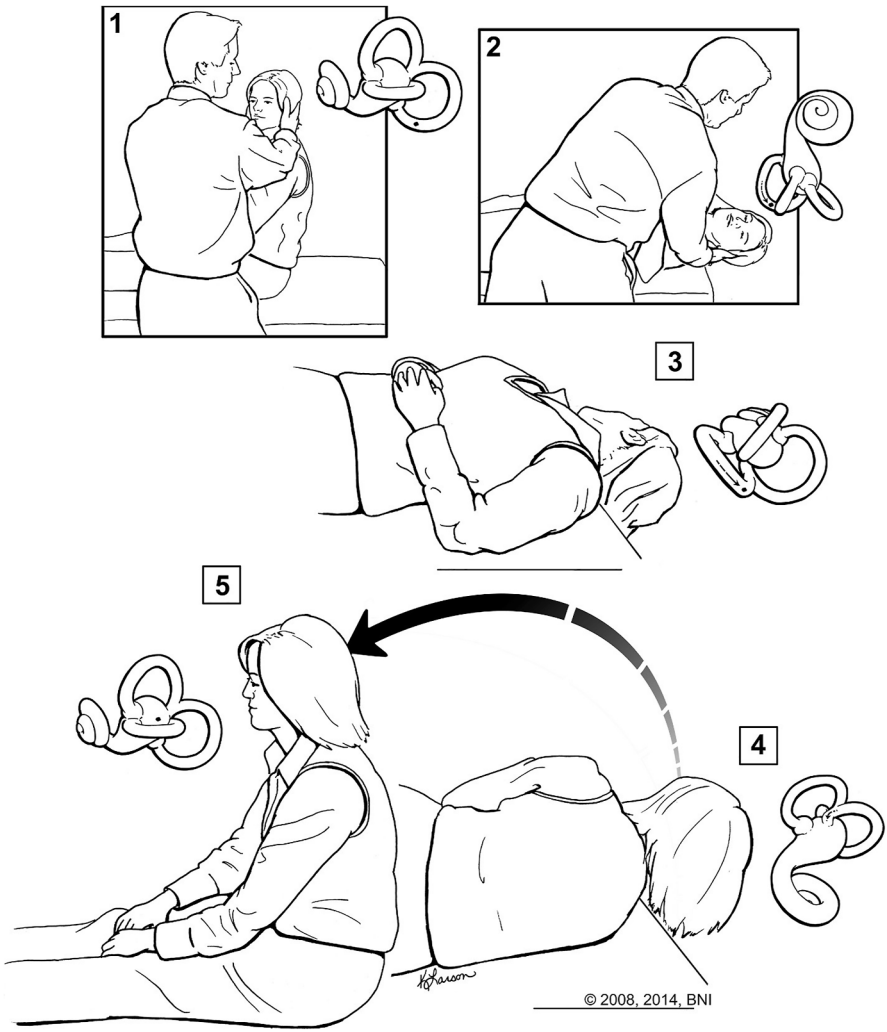

Fig. 12. Epley maneuver. (*Used with permission from* Barrow Neurological Institute, Phoenix, Arizona.)

reposition displaced otolith from the canal back to the utricle (**Fig. 12**, Video 6). *The treatment for posterior canal BPPV has 90% effectiveness, with a number needed to treat of 1.6.*[59] For every 3 patients with posterior canal BPPV, 2 will have complete improvement.[59] This is one of the most effective treatments in all of medicine, but it assumes a correct diagnosis. BPPV untreated with an Epley has a 46% recurrence risk versus 20%,[9] 63.3% had lost working days, 4.6% had changed their jobs, and 5.7% had quit their jobs, due to vertigo symptoms.[60] Long-term treatment with meclizine or other vestibular suppressants is not recommended.[61] The American Geriatric Society specifically recommends against the use of meclizine in elderly patients due to their anticholinergic side effects.[62] One study found that the use of first-generation antihistamines (such as meclizine), which cross the blood-brain barrier, doubles the rate of fractures in elderly patients.[63] Furthermore, no medication treats the underlying

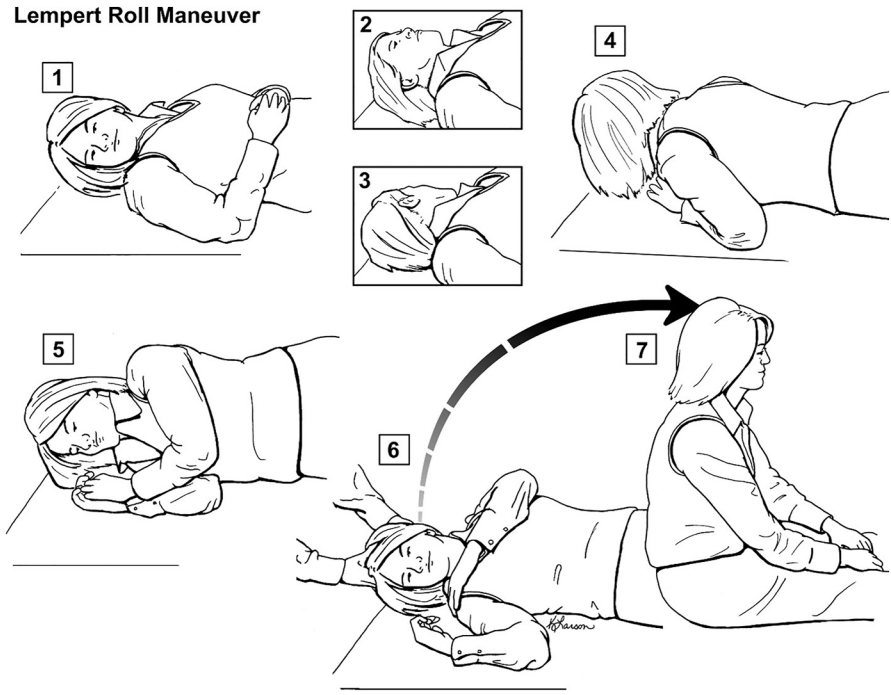

Fig. 13. Barbeque roll maneuver.

pathology in BPPV; canalith repositioning maneuvers do. Some patients started on medications for their dizziness end up taking the medicine for long periods of time, often without a specific diagnosis.[64,65] All of that said, in patients who are very symptomatic due to ongoing dizziness, acute treatment in the ED or for 2 to 3 days following discharge can be used for symptom relief. In this setting, one study found that antihistamines were superior to benzodiazepines.[66]

Horizontal canal benign paroxysmal positional vertigo. The treatment for horizontal canal BPPV is called the Lempert 360 roll maneuver/BBQ Roll test (**Fig. 13**, Video 7). Another technique called forced prolonged positioning where patients sleep on the unaffected ear for many hours will also resolve symptoms. About 90% of cases resolve in 1 week and all resolve within 4 weeks.[59]

Vestibular neuritis. MRI for typical vestibular neuritis is not indicated. Steroids are often prescribed for patients with neuritis although evidence of its efficacy is extremely limited.[67] These patients should follow-up with vestibular rehab, physical therapy, audiology, ENT, neurology, or neurotology based local availability.

Current controversies. While HINTS was validated for neuro-otologists, studies show that trained emergency physicians can perform the HIT with similar accuracy.[21,68] Training EM physicians in performing HIT and performing a systematic dizziness evaluation has been shown to be effective.[69–72] Gerlier and colleagues studied the effectiveness of educational intervention for BPPV. They showed a standardized educational intervention in the ED shortened length of stay, increased use of canalith

repositioning and improved clinician satisfaction which correlated with effectiveness of their clinical management.[73]

FUTURE CONSIDERATIONS

TriAGE+ score is a scoring system that uses history instead of physical examination findings. The original study had a sensitivity of 96.4% (87.3–99.5) and a specificity of 99.8% (98–100) in a Hong Kong patient population.[74] A recent article looking at that score, however, showed only a sensitivity of 26.5% (18.2–36.1) but a specificity of 96.3% (93.5–98.1) suggesting it can be used to rule in a central cause.[68]

Sudbury Vertigo Risk Score similarly uses a mix of history and physical tool that has a sensitivity of serious diagnosis of 100% (95% CI: 96.5%–100%) and a specificity of 69.2% (95% CI: 67.8%–70.5%) for a score less than 5. It has 7 parts that contribute to the score, male, age greater than 65, diabetes, hypertension, neurologic deficits (diplopia, dysarthria, dysphagia, dysmetria, ataxia), motor/sensory, cerebellar, BPPV. These scores show promise if validated.[75,76]

This work on creating an ED EYE phone app to allow for a more efficient way to diagnose and consult a tele-dizzy program without expensive video-oculography. Finally, research is ongoing to test the importance of using Frenzel lenses.

SUMMARY

Using a systematic approach to dizziness similar to what we do for other diseases allows physicians to confidently make diagnoses that lead to correct treatment and should reduce misdiagnosis of cerebrovascular events and overuse of unnecessary testing. Learning to perform HINTS and diagnostic and therapeutic bedside maneuvers for BPPV has been shown to be possible in by emergency medicine physicians. If uncertain or untrained, always error on the side of caution. Patients who cannot walk independently should be assumed to have central causes. CT scans are unnecessary and add expense and can lead to false reassurance and even early MRI can be falsely negative.

CLINICS CARE POINTS

- Use a "timing and triggers" approach to the diagnosis of dizziness.
- Each category (acute vestibular syndrome [AVS], spontaneous episodic vestibular syndrome, and triggered episodic vestibular syndrome) is tightly associated with a specific set of examination maneuvers and diagnoses.
- Do not perform a HINTS examination in patients with an episodic vestibular syndrome.
- Do not perform a Dix-Hall Pike or Supine Roll test in patients with an AVS.
- Learn to use evidence-based physical examination and therapeutic techniques to improve diagnostic accuracy and treatment of benign paroxysmal positional vertigo.
- The 95% of AVS patients have either neuritis (vestibular neuritis or labyrinthitis) or posterior stroke. A rare but important cause is Wernicke encephalitis (thiamine deficiency).

DISCLOSURE

The authors have nothing to disclose.

SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.emc.2025.08.006.

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